REMARKS

1. General Matters

1.1. A final rejection was mailed on September 12, 2005. In response, on December 8, 2005, Applicants filed an amendment after final rejection.

Subsequently, applicants received an advisory action mailed February 28, 2006. The December 8, 2005 amendment was not entered, for two reasons:

(1) the examiner questioned whether there was supporting "written description" for claim 1 as amended; and

(2) new claims 53-56 were considered drawn to non-elected inventions.

Applicants have filed an RCE on even date herewith, requesting entry of the instant amendment. The instant amendment differs from the one filed December 8, 2005, primarily in that

(1) it sets forth the written description basis for amended claim 1, see section 2.3 below; and

(2) it omits the previously proposed new claims 53-56 (there is a new claim 53 unrelated to the December 8 one).

Since it presents the same amendment to claim 1 as was refused entry previously, the first action after the RCE, if a rejection, cannot be made "final".

1.2. We take this opportunity to remind the Examiner that the final rejection only objected to claims 20, 25, 35, 44, 45 and 47-49, i.e., the Examiner has conceded that patentable subject matter is present.

1.3. The Declaration of Else Tønnesen was filed under 37 CFR 1.132, as is evident from the fact that it makes no statements regarding the date of invention.

2. Non-Art Issues

2.1. The "written description" rejection of claims 37 and 38 is moot as those claims have been cancelled. This also moots the objection to the improper Markush group of claim 38.

2.2. The objection to claims 28 and 36 as having the same scope is moot as claim 36 has been cancelled.

2.3. Claim 1 has been amended so it refers to "treatment or prophylaxis of acute inflammation of the lung or airways" rather than to "treatment or prophylaxis of a non-ischemic condition characterized by acute inflammation of the lung or airways". The advisory action said that this raised the issue of new matter/written description.

Claim 1, as amended, refers to treatment of a symptom (acute inflammation) of a bodily disorder, whereas original claim 1 referred to treatment of a bodily disorder (a non-ischemic condition characterized by acute inflammation).

A careful review of the specification reveals that, in treating the non-ischemic condition, we usually were not expecting to cure the condition. Rather, the goal was to ameliorate the symptom, the acute inflammation associated with the condition.

Page 1, lines 9-13 states "the present invention relates to a method for treatment or prevention of inflammation in a non-ischemic condition... In particular, the invention relates to the treatment of inflammation under non-ischemic conditions".

Next, at page 4, lines 16-19, the specification asserts that "the effect of treatment... was established in models of lung inflammation, systemic inflammation, kidney inflammation, and diseases of the urinary tract".

At line 24, we find the heading "acute/subacute LPS-inhalation induced lung and airway inflammation".

Inflammation is associated with neutrophil and eosinophil activity, see page 5, lines 9-11, and at page 4, lines 25-26, we

assert "treatment with the compounds significantly prevented the neutrophil and eosinophil airway and lung infiltration".

We then proceeded to discuss chronic obstructive pulmonary disease. COPD is caused by an inflammatory condition, P5, L14-34, so treatment of the inflammation is curative as well as ameliorative.

Page 9, lines 22-23 says, "the present invention relates to a method for treatment or prevention of an inflammatory condition".

The condition being "non-ischemic" is identified as being a "preferred embodiment" (P12, L18-20). It thus is not necessary that claim 1 make reference to a non-ischemic condition. However, such reference <u>is</u> made in new claim 53.

The term "inflammatory condition" is defined at P10, L1-7, and contrasted with the "inflammatory diseases" of P10, L9-P12, L16. In essence, an "inflammatory condition" causes recruitment of inflammatory cells (resulting in inflammation), and such inflammation may give rise to an "inflammatory disease".

P34, L12-19 reports that the claimed combined treatment "almost completely prevented the influx of inflammatory cells in lung tissue exposed with LPS". The person skilled in the art would recognize that this meant that applicants were in possession of a method of preventing LPS-induced lung inflammation.

2.4. Since claim 1 no longer refers to a "condition", claims 20, 23 and 25 have been reworded.

2.5. Claims 41 and 42 have been amended so that they parallel claims 28 and 30, and are properly limited by dependent claims 47-49.

2.6. New claims 51 and 52 parallel claim 45, but depend from 28 and 41 respectively.

3. Prior Art Issues

3.1. Claims 1, 2, 5, 23 and 39 stand rejected as anticipated by Akamatsu. In the last paragraph on page 3, the Examiner states:

> Applicant's arguments have been fully considered but are not deemed persuasive. The instant claims do not state **"a method for the treatment or prophylaxis of acute inflammation of the lung or airway"** (Emphasis added). The instant claims are broadly drawn to "a method for the treatment or prophylaxis of a nonischemic condition, **characterized by acute inflammation of the lung or airway"** (Emphasis added).

As a result of the present amendment, claim 1 now is drawn to "a method for the treatment or prophylaxis of acute inflammation of the lung or airway". In contrast, Akamatsu discloses alleviation of anemia. We believe that the amendment of claim 1 is following an examiner's suggestion of patentable subject matter.

3.2. Claims 1, 26-30, 36, 40-42, 46 and 50 stand rejected as obvious over Akamatsu in view of Delgado Hernandez. The amendment to claim 1 should affect this rejection, as well as the one for anticipation.

Akamatsu *et al.* describes the treatment of anemia, which cannot be considered as a condition characterized by acute inflammation of the lung and airways.

Hernandez Delgado et al. shows that alpha-MSH may have an effect on myeloperoxidase in LPS-induced lung inflammation, which suggests that alpha-MSH treatment may have an effect that could be associated with inhibition of neutrophils. However, the paper provides no data supporting that EPO could have anti-inflammatory effects in inflammatory lung disease.

None of the prior art documents disclose or suggest that EPO in combination with alpha-MSH treats or prevents acute

inflammation of the lung or airways. Therefore, there is no incentive for the person skilled in the art to combine Akamatsu *el al.* with Hernandez *et al.*

3.3. The objection to claims 20, 25, 35, 44, 45, 47-49 as dependent on a rejected claim should be moot in view of the amendments to base claim 1, which overcome the rejections.

Respectfully submitted,

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